CONCLUSIONS

This analysis evaluated disease symptoms as related to smoking, consumption of alcohol, exposure to TSP and SO_2 outdoors, diet, age and earnings in 1973 as a proxy for socio-economic status. The study found that the only statistically significant relationship for air pollutants, which had the expected signs, were between TSP and cough and coronary heart attack and between SO_2 and chest pain. A slightly less significant relationship was found between TSP and shortness of breath.

The most significant "explanatory variables" for respiratory symptoms were dieting, smoking, alcohol consumption, socio-economic status, and air pollution. In this context, a positive relationship was found between shortness of breath and dieting, smoking, TSP concentrations, and one of the alcohol consumption variables. SO_2 and earnings were found to negatively effect shortness of breath. Dieting, age, earnings, and to a lesser extent SO_2 had negative effects on coughing while smoking, alcohol consumption and TSP had positive effects on the symptom.

The need to diet and smoking were consistently found to be positively correlated and economic status negatively correlated with cardiovascular system problems. Significant positive relationships between alcohol consumption and cardiovascular problems were found for chest pain and to a lesser extent coronary heart attack. Age was found to be negatively correlated with the occurance of all cardiovascular symptoms. However, a significant relationship between age and a symptom was only found for coronary heart attach. TSP was found to have a significantly positive effect on the incidence of coronary heart failure while SO_2 was found to positively effect chest pain. SO_2 was found to have a negatively significant effect on coronary heart attack. Finally, no air pollution variables were found to significantly influence severe chest pain. These findings suggest that the air pollution variables may be "masking" or replacing some other significant affects. Only similar analyses will perhaps lead to a net effect on ambient air quality on certain disease symptoms.

The list of symptoms were collected from the 1967-68 period while air pollution data were recorded for the year 1977, by zip code. Thus, only a weak inference can be made between air pollution common to times and symptoms. Because of time and manpower limitations, past air pollution data have not been included, inclusive of where the twin resided since 1945. Thus, unless the twin resided in the same place and there were no substantial changes in ambient air quality between the 1960's and late 1970's, the link between exposure and symptom can occur only be chance. Future research should center on more closely aligning symptoms with similar locations of exposure.

Evaluation of ordinary least squares and a more advanced technique of econometric analysis called "probit" yielded almost identical results except for a "scale" factor on the coefficients over at least fourty variants of

the preliminary model. This leads us to believe that OLS may be a reasonable technique to apply to more "robust" variables and theoretical systems.

Adequate variables measuring total inhalation of particulate, diet in terms of fat consumption, and "stress" variables have not been modelled. It is unlikely that current consumption of cigarettes, alcohol consumption as measured by a weighted sum of pure alcohol, or the need to diet, accurately reflect the impact on body processes. For example, a "heavy" smoker may have quit smoking in the early 1960's and yet retain some of the respiratory symptoms. Until these variables are adequately measured by complete exposure, it is unlikely that they will be useful for interpretation or prediction for policy purposes.

The effects of air pollution on health symptoms found in this study are roughly consistent with earlier work. However, with minor exception, all earlier sutdies focused on the effects of air pollution on mortality and morbidity. In four separate studies, Lave and Seskin (20)(21)(22)(23) McDonald/Schwing (24), Crocker (25), and Liu/Yu (26) all found partial linkages between air pollution and mortality and morbidity. Ostro (27) estimated the effects of total suspended particulate on work loss days. A comparison of the Ostro and Crocker et.al. results to the results presented in this study revealed that estimates presented in this study, as predicted, are of smaller magnitudes. Only Page (28) used a methodology remotely similar to the symptom-pollution relationships analyzed in this study. Page's measure of health effects was a self reported diary from 1,000 victims of respiratory illness as to whether they felt better, worse, or the same.

In order to derive total savings in health care costs, a 30 percent improvement in ambient air quality was assumed. The societal prevalence and death rates for nine diseases were used as proxys for the probability of incurring a disease or death given the presence of a symptom in the sample population. In this context, estimates of cost savings for a 30 percent reduction in maximum 24 hour ambient concentration of TSP and $S0_2$ was estimated to be over $4 million in males 55 to 65 years of age. Extrapolation of these savings to the total U.S. population yields an estimate of health cost savings of nearly $100 million.

APPENDIX 1

METHODOLOGY USED FOR FOOD CONVERSIONS

Table 17 presents the figures used to calculate the yearly consumption of different nutrients for the questionnaire respondents. In order to calculate Table 17, several assumptions were made on the serving sizes, given a questionnaire response. These assumptions, along with the figures in Table 19 were used to estimate Table 17. Figures in Table 19 were gathered from alternate sources (29)(30)(31)(32).

The following procedure was used to calculate nutrients ingested per year from consuming pasteries and candies:

(1) if more than one response was given the sample was deleted, and

(2) if only one response was given then the following was assumed:

Response	Assumption
0 never	0 serving/day
1 several times a day	3 servings/day
3 once a day	1 serving/day
5 less often	.5 serving/day

Nutrients in pork, frankfurters, beef, cereal, eggs, fish, vegetables and fruit were determined via the following procedure.

(1) if more than one response was given the sample was deleted, and

(2) if only one response was given then the following was assumed:

Response	Assumption
0 never	0 servings/day
1 daily	1 serving/day
3 once or twice/week	6 servings/month
5 once or twice/week	1.5 servings/month
7 less often	6 servings/year

For example, to determine the grams of protein consumed from eating a serving of frankfurters daily, multiply the 7 grams/day from Table 17 by 365 days in the year, i.e.,

$$7 \text{ gr/day} \cdot 365 \text{ days/year} = 2555 \text{ gr/year}$$

which gives the yearly consumption of protein from consuming frankfurters daily. If the respondent answered that he consumed frankfurters once or twice a month, it was assumed they consumed 1.5 servings per month. Therefore

the equation to calculate the grams of protein ingested in a year is

1.5 servings/month.7 gr/serving .12 months/year = 126 gr/year.

The yearly consumption of a nutrient for each respondent may be calculated by summing over the types of food for each nutrient. The yearly figures were used in the regression analysis to determine the importance of these nutrients to different symptoms reported.

TABLE 6.17 FIGURES USED TO CALCULATE THE YEARLY CONSUMPTION OF DIFFERENT NUTRIENTS FOR THE QUESTIONNAIRE RESPONDENTS BY TYPE OF FOOD CONSUMED AND TYPE OF RESPONSE WHERE APPROPRIATE[1]

Nutrient / Type of Food/var #	Protein (gm)	Fats (gm)	Fatty Acids uns (gm)	Fatty Acids sat (gm)	Carbohydrates sugar (gm)	Carbohydrates fiber (gm)	Vit. A (iu)	Ribo-flavin (mg)	Niacin (mg)	Thiamin (mg)	Calcium (mg)	Iron (mg)
Pasteries[2]												
(51) 1	5475	16425	10950	5475	32850	0	219000	109.5	547.5	54.75	36135	657
3	1825	5475	3650	1825	10950	0	73000	36.5	182.8	18.25	12045	219
5	912.5	2737.5	1825	912.5	5475	0	36503.65	18.25	91.25	9.13	6025.5	109.5
Candy[2]												
(52) 1	17520	19710	3285	5475	35040	0	175200	219	219	43.8	328.5	657
3	1460	6570	1095	1825	11680	0	58400	73	73	14.6	109.5	219
5	730	3285	547.5	912.5	5840	0	29200	36.5	36.5	7.3	54.75	109.5
Bread White (53)	$X_3 \cdot 730$	$X_3 \cdot 365$	na	na	$X_3 \cdot 5091.75$	$X_3 \cdot 18.25$	-	$X_3 \cdot 21.9$	$X_3 \cdot 255.5$	$X_3 \cdot 25.55$	$X_3 \cdot 8760$	$X_3 \cdot 255.5$
Whole Milk (54)	$X_4 \cdot 3285$	$X_4 \cdot 3285$	$X_4 \cdot 1095$	$X_4 \cdot 1825$	$X_4 \cdot 4380$	0	$X_4 \cdot 127750$	$X_4 \cdot 149.65$	$X_4 \cdot 73$	$X_4 \cdot 25.55$	$X_4 \cdot 105120$	$X_4 \cdot 36.5$
Skim Milk (55)	$X_5 \cdot 3285$	-	-	-	$X_5 \cdot 4380$	0	$X_5 \cdot 3650$	$X_5 \cdot .44$	$X_5 \cdot .73$	$X_5 \cdot 32.85$	$X_5 \cdot 108040$	$X_5 \cdot 36.5$
Coffee (56)	$X_6 \cdot 109.5$	$X_6 \cdot 36.5$	na	na	$X_6 \cdot 292$	0	0	$X_6 \cdot 36.5$	$X_6 \cdot 328.5$	$X_6 \cdot 3.65$	$X_6 \cdot 1679$	$X_6 \cdot 83.95$
Coffee w/ tsp. sugar (57)	$X_7 \cdot 109.5$	$X_7 \cdot 36.5$	na	na	$X_7 \cdot 4307$	0	0	$X_7 \cdot 36.5$	$X_7 \cdot 328.5$	$X_7 \cdot 3.65$	$X_7 \cdot 1679$	$X_7 \cdot 83.95$
Tea (58)	$X_8 \cdot 36.5$	0	na	na	$X_8 \cdot 328.5$	0	0	$X_8 \cdot 14.6$	$X_8 \cdot 328.5$	0	$X_8 \cdot 1825$	$X_8 \cdot 73$
Tea w/ tsp. sugar (59)	$X_9 \cdot 36.5$	0	na	na	$X_9 \cdot 4343.5$	0	0	$X_9 \cdot 14.6$	$X_9 \cdot 328.5$	0	$X_9 \cdot 1825$	$X_9 \cdot 73$

TABLE 15 (continued)

Pork[3]												
(60) 1	7300	8760	4380	3825	0	0	o	80.30	1715.50	284.70	3825	985.5
3	1440	864	432	324	0	0	0	7.92	169.20	28.80	324	97.2
5	360	432	216	162	0	0	0	3.96	84.60	14.04	162	48.6
7	120	144	72	54	0	0	0	1.32	28.20	4.68	54	16.2
Frankfurters[3]												
(61) 1	2555	5475	na	na	365	0	na	40.15	511. C	292	1095	292
3	252	540	na	na	36	0	na	3.96	50.4	28.8	108	28.8
5	126	270	na	na	18	0	na	1.98	25.2	14.4	54	14.4
7	42	90	na	na	6	0	na	.66	8.4	4.8	18	4.8
Beef[3]												
(62) 1	7300	9855	4745	4745	0	0	18250	58.40	1460	18.25	3825	912.5
3	1440	972	468	468	0	0	1800	5.76	144	1.8	324	90
5	360	486	234	234	0	0	900	2.88	72	.9	162	45
7	120	162	78	78	0	0	300	.96	24	.3	54	15
Cereal[3]												
(63) 1	730	-	na	na	7665	0	0	7.38	182.5	40.15	1460	146
3	72	-	na	na	756	0	0	.72	18.0	3.96	144	144
5	36	-	na	na	378	0	0	.36	9.0	1.98	72	7.2
7	12		na	na	1.26	0	0	.12	3.0	.66	24	2.4
Eggs[3]												
(64) 1	4380	4380	2190	14 60			430700	109.5	-	36.50	19710	803
3	432	432	216	144		-	42480	10.8	-	3.6	1944	79.2
5	216	216	108	72			21240	5.4	-	1.8	972	39.6
7	72	72	36	24			7080	1.8	-	.6	324	13.2
Fish[3]												
(65) 1	6205	1825	365	365	1825	o	na	21.9	985.5	10.95	12410	365
3	612	180	36	36	180	0	na	2.16	97.2	1.08	1224	36
5	306	90	18	18	90	0	na	1.08	48.6	.54	612	18
7	102	30	6	6	30	0	na	.36	16.2	.18	204	6
Vegetables[3]												
(66) 1	1095		na	na	8103	292		18.25	730	47.45	3650	292
3	108		na	na	799.2	28.8		1.80	72	4.68	360	28.8
5	54		na	na	399.6	14.4		.90	36	2.34	180	14.4
7	18	-	na	na	13).2	4.8		.30	12	.78	60	4.8

TABLE 6.17 (continued)

Fruit[3]												
((,7) 1	-	-	na	na	5840	730	18250	7.30	36.5	14.60	2920	146
3	-	-	na	na	576	72	1800	.72	3.6	1.44	288	14.4
5	-	-	na	na	288	36	900	.36	1.8	.72	144	7.2
7	-	-	na	na	96	12	300	.12	.6	.24	48	2.4

Footnotes: (1) There are two types of figures here, Var. 51, 52 and 60-67 already have the questionnaire response included within the calculation and only need to he identified by response. Var. 53-59 do not have response included in the calculation and therefore Lhe coefficient must be multiplied by Lhe response.

(2) If more than two responses where given on Lhe questionnaire then these samples were deleted. If this is not the case, the following was assumed.

Response	Assumption
0 never	1) servings/day
1 several times a day	3 servings/day
3 once a day	1 servings/day
5 less often	.5 servings/day

(3) Again if more than one response was given the sample was dropped and the following assumptions were made for the samples used.

Response	Assumption
0 never	0 servings/day
1 daily	1 servings/day
3 once or twice a week	6 servings /month
5 once or twice a month	1.5 servings/month
7 less often	6 servings/year

Notes: na: suitable data was notavailable but I he nu trient is suspected LO be present
- : only a trace has been detected
0 : the nutrient is not present and is I1oL suspected to be so

References: 1. Hamilton, E.M. and E. Whitney, Nutrition: Concepts and Controversy
2. Nutrition Search Co., Nutrition Almanac, McGraw Hill Book CO., 1975
3. National Dairy Council, Guide LO Good Eating, 1980

TABLE 6.18 FIGURES USED TO CALCULATE YEARLY CONSUMPTION OF NITROSAMINES BY QUESTIONNAIRE RESPONDENTS BY TYPE OF FOOD CONSUMED AND QUESTIONNAIRE RESPONSE

Type of Food	(Var. #)	Response	Nitrosamines (µg)
Pork	60	1	31.03
		3	3.06
		5	1.53
		7	.51
Frankfurters	61	1	224.84
		3	22.18
		5	11.09
		7	3.70
Beef	62	1	na
		3	na
		5	na
		7	na
Fish	65	1	31.03
		3	3.06
		5	1.53
		7	.51

Note: Minimum values are used here

References: Unpublished manuscript by Ron Shank for EPA Nitrates report

TABLE 6.19 LEVELS OF NUTRIENTS AND NITROSAMINES PER SERVING BY TYPE OF FOOD

Nutrients / Type of Food	Serving	Protein (gm)	Fats (gm)	Fatty Acid uns (gm)	Fatty Acid sat gm)	Carbohydrates sugar (gm)	Carbohydrates fiber (gm)	Vit. A (iu)	Riboflavin (mg)	Niacin (mg)	Thiamin (gin)	Calcium (ma)	Iron (mg)	Nitrosamines (µg)
Pasteries (51)	1 avg+	5	15	10	5	30	0	200	.10	.5	.05	33	6	0
Candy Milk Choc (52)	2 oz+ bar	4	18	3	5	32	0	160	.2	.2	.04	30	.6	0
Bread White (53)	1 slice* 22 slice/ loaf	2	1	na	na	13.95	.05		.06	.7	.07	24	.7	0
Whole Milk (54)	1 glass*	9	9	3	5	12	0	350	.41	.2	.07	288	.1	0
Skim Milk (55)	1 glass*	9	-	na	na	12	0	10	.44	.2	.09	296	.1	0
Cof fee (56)	1 cup*	.3	.1	na	na	.8	0	0	.01	.9	.01	4.6	.23	0
Coffee WI tsp. sugar (57)	1 cup w/1 tsp. sugar	.3	.1	oa	na	11.8	0	0	.01	.9	.01	4.6	.23	0
Tea (58)	1 cup*	.1	-	na	na	.9	0	0	.04	.1	0	5.0	.20	0
Tea w/ tsp. sugar (58)	1 cup w/1 tsp. sugar	.1	-	na	na	11.9	0	0	.04	.1	0	5.0	.20	0
Pork (60)	3 oz≠	20	24	12	9	0	0	0	.22	4.7	.78	9	2.7	.085
Beef (62)	3 Oz≠	20	27	13	13	0	0	50	.16	4.0	.05	9	2.5	na

TABLE 17 (continued)

Frankfurters (61)	2 oz≠	7	15	na	na	1	0	na	.11	1.4	.8	3	.8	.616
Cereal Cornflakes (63)	1 Cnp no sugar	2		na	na	21	0	0	.02	.5	.11	4	.4	0
Eggs (64)	2	12	12	6	4		0	1180	.3		.10	54	!.2	0
Fish Haddock (65)	3 Oz≠	17	5	3	1	5	0	na	.06	2.7	.03	31	1	.085
Vegetables (66)	1 cup	3		na	na	22.2	.8		.05	2.0	.13	10	.8	0
Fruit-apple (67)	1 med			na	na	16	2	50	.02	.1	.04	8	.4	.0

Footnotes: (1) all figures came from reference (1) except for those which came from reference (2).

Notes: * - These foods are measured in same manner as in questionnaire
≠ - Daily recommended servings are not used here as both references 1 and 2 used 3 oz. as an average serving
+ - Given there are no dally recommended servings for these variables. We assumed the average serving of pastery as 1 and an average serving of candy as a candy bar

preferences:
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3. Miller, M., and L. Leaven, Anatomy and Physiology, 16th edition, Macmillan Publishing Co., Inc., 1972. Especially Chapter 19.

4. A complete discussion of the NAS-NRC Twin Registry can be found in Zdenek Hrubec and James V. Neel, "The National Academy of Sciences - National Research Council Twin Registry: Ten Years of Operation," in Twin Research: Biology and Epidemiology, New York: Alan R. Teis, 1978.

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6. Zygosity is classified as either monozygotic (MZ) for identical twins and dizygotic (DZ) for fraternal twins.

7. U.S. Environmental Protection Agency, SAROAD: Information, Research Triangle Park, North Carolina, February 1979.

8. See the various reports from the United States Surgen General's office on the effects of smoking and health.

9* See, for example, Gould, Lawrence, "Cardiac Effects of Alcohol," American Heart Journal, volume 74, January-March 1970.

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18. Ostro, Bart D., "The Effects of Air Pollution on Work Loss and Morbidity," submitted to the Journal of Environmental Economics and Management, 1982.

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Chapter VII

ANALYTICAL PRIORS AND THE SELECTION OF AN "IDEAL" AIR POLLUTION EPIDEMIOLOGY DATA SET

INTRODUCTION

Widespread concern with the health effects of economics benefits generated by air pollution control programs has provoked a number of statistical studies of the association between air pollution and health status. However, the appropriateness of methodology and accuracy of the results of these studies have been widely disputed. The purposes of this paper, therefore, are threefold. First, we examine the role of optimal decision rules in testing the validity of price information to produce "best" estimates of the human health losses attributable to air pollution and the economic valuation of these losses. Secondly, we examine the use of price-information decision rules in previous air pollution-human health studies. Finally, based on optimal decision rules, we summarize statistically accepted prior information about the elements of an "ideal" air pollution epidemiology data set.

Statistical estimation of the degradation of health due to air pollution and the economic valuation thereof requires the use of prior information decision rules in four principal areas: (1) model selection (e.g., simultaneous, recursive, errors in variables, or single equations); (2) choice of functional form and the dimension of the design matrix; (i.e., matrix of exogenous variables); (3) the choice of values assigned to each element of the design matrix, if under the control of the experimenter; and (4) choice of the density function of the dependent variable. Most statistical analysis involved regressing a dependent variable (usually mortality and morbidity rates on time-to-failure for a system) a set of covariates which have been postulated to explain the variation in the dependent variable. Imposing prior information through exact parametric restrictions (whether correct or not) reduces the variance of estimated parameters. However, if incorrect, the restrictions increase estimator bias. Thus , the use of prior information, which is always incorrect to same degree except by chance, necessarily involves a tradeoff between the bias and efficiency of estimated parameters.

We evaluate this tradeoff in terms of the risk, i.e., the expected loss associated with each estimated parameter, measuring loss as the squared error of each estimated parameter relative to its true value, risk equals the sum of estimated parameter variances and squared biases. Stated somewhat differently, the researcher must choose decision rules which maximize the net benefit from utilizing prior information, where the benefit of such action is the resulting variance reduction and the cost is the resulting increase in bias. He seeks a middle ground somewhere between the overly restrictive case (high bias, low variance) and the totally unrestrictive case (unbiased, high variance).

In seeking decision rules for imposing prior information which minimize risk, there are valuable guidelines for accepting or rejecting hypotheses of exact prior restrictions (the most common type) and inequality restrictions. Regardless of the correctness of equality restrictions the positive-part Stein-rule estimator introduced by Baranchik (1964) which possesses minimum risk compared to the unrestricted estimator or the pre-test estimator (based on the standard decision rule to accept or reject the null hypothesis at a pre-specified level of significance). In addition, if inequality restrictions are correct in sign, they always exhibit less risk than the unrestricted estimator [see Judge, et al., 1980].

Our general conclusion regarding previous analysis of the effects of air pollution on human health and the valuation of these impacts. is that the preponderence of attempts to impose prior information have failed to minimize risk. Weak priors have rarely been correctly (if at all) tested before being imposed, while other strong but untestable priors have been ignored. We also conclude that the ideal data set, based on optimal decision rules, is not comprised of an exhaustive set of explanatory variables, since this would lead to unacceptably large estimator variances. Conversely, the ideal data set does not consist of a design matrix which excludes potentially important explanatory variables previous to statistical testing. To the extent that magnitudes of explanatory variables are under the control of the experimenter, the values assigned to an ideal data set should minimize risk subject to a given experiment budget constraint. If variables are not under the experimenter's control, the composition of the design matrix should be determined by optimal statistical tests based on prior information. An ideal data set can only be defined in conjunction with such information.

The plan for the remainder of the paper is to examine optimal decision rules for the use of prior information in section II and, in light of this, provide a critical review of the epidemiological literature measuring the effects of air pollution on human mortality and morbidity in section 111. A similar review of the literature which attempts to value these adverse health

affects is presented in section IV. Based on statistically accepted priors, in section V we suggest superior data sets for potential analysis. Finally, conclusions about optimal use of prior information are drawn in section VI.

USE OF PRIOR INFORMATION

Statistical estimation of the effects of air pollution on human health is impossible without the use of some prior information. This may take the form of model selection, choice of function form and dimension of the design matrix, selection of the values of each element of the design matrix (for variables under control of the experimenter), and choice of the density function for the dependent variable. The imposition of prior restrictions in these areas leads to an increase in the efficiency of estimated parameters. However, if restrictions are incorrect, estimated parameters are biased [see Judge, et al., (1980, ch. 11)]. Thus, the inescapable act of imposing prior information requires that the econometric researcher walk a tightrope between efficiency, on the one hand, and bias, on the other.

We proceed, therefore, to seek information regarding the optimal use of prior information which minimizes risk. In the context of regression analysis, we first define loss as the cost incurred if our estimate of the true value of the parameter vector of β is $\hat{\beta}$. Adopting a squared error loss criterion, we may write loss as

$$L = (\underline{\beta}-\underline{\beta})'(\underline{\hat{\beta}}-\underline{\beta}), \quad (1)$$

involving the k-dimensional vectors β and f3. Risk is defined as the expected value of loss:

$$P = E\,[(\hat{6}-6)'(\hat{6}-6)], \quad (2)$$

which equals the sum of variances for each element of $\hat{\beta}$ plus the sum of squared biases for each element of $\hat{\beta}$. Our objective is to minimize the risk from imposing prior restrictions.

Choice of Functional Form and Dimension of the Desire Matrix

We first consider this objective for the choice of functional form and dimension of the design matrix within the context of the testing of nested hypotheses[1/] for a single equation regression model. Four types of prior information may be imposed: exact restrictions, stochastic restrictions, inequality restrictions, and prior density functions. We compare the risks of utilizing these types of prior information to that of the unrestricted estimator, the pre-test estimator, and the Stein-rule estimator. The pre-test

estimator is simply the standard nested hypotheses test procedure whereby the null hypothesis (generally $\beta = 0$) is accepted or respected based on some predetermined level of significance. One example of a pre-test estimator is accepting or rejecting nested models of the quadratic Box-Cox (1964) form based on pre-determined levels of the likelihood function. Restrictions on estimated parameters lead to the inverse semi-log, semi-log, translog, generalized linear, quadratic, generalized square root quadratic, and linear models. [See Berndt and Khaled (1979)]. Choice among these nested models is typically based on the likelihood ratio test statistic. Additional restrictions allow testing of hypotheses about consumer behavior (homotheticity, additivity, and symmetry) or cost, production and profit function (homotheticity, homogeneity).

Exact information is the most common type of prior restriction. If the exact prior information is correct, the restricted least squares estimates are "best" estimates (i.e., minimum variance, unbiased). Incorrect exact prior restrictions, however, lead to biased estimates, which have smaller variances than under the correct model. The risk for the restricted least-squares estimator increases monotonically and exceeds the constant risk of the unrestricted maximum likelihood estimator, (MLE) over a wide range of hypothesis error under the assumptions of the general linear model. Further, the pre-test estimator has greater risk than the MLE estimator over a wide range of hypothesis error and hence, is inadmissible under our risk function criterion.

Stein-rule estimators [see Judge, et al. (1980, pp. 432) and Judge and Bock (1978)] exhibit less risk over the entire parameter space than the unrestricted and restricted MLE estimators, and the pre-test estimator. The positive-part Stein-rule estimator involves testing the hypothesis that $\beta_o = 0$, where β_0 is a vector of K_2 parameters. If $u_{(k_2)}$, the value of the likelihood ratio statistic, is less than or equal to $C_{(k_2)}$, where

$$C_{o_{(k_2)}} < C^*_{(k_2)} < 2C_{o_{(k)_2}} \text{ and } C_{o_{(k_2)}} = F_{(k-2),(T-k)}/k(T-k+2),$$

where k is the total number of variables and T is the total number of observations, we exclude the k_2 variables from the model. Otherwise, we employ the Stein-rule to transform the unrestricted MLE estimates using $C_*(k_2)$ and $u_{(k_2)}$ [see Judge et al. (1980)]. A second type of prior

information involves the use of stochastic prior information. Restrictions are assumed to hold subject to a normally distributed random vector. The sampling results for this type of prior restriction are parallel to those for the equality restricted estimator [see Judge et al. (1980)]. Inequality constraints comprise a third type of restriction. The risk function for the inequality constraint (when the direction is correct) is less than or equal to that of the MLE over the whole range of the parameter space the risk of the inequality pretest estimator (again when the direction is correct) is less than that of the traditional pretest estimator over almost the entire parameter space [see Judge and Yancky (1978)]. This result, which is particularly powerful, has largely been ignored by applied econometricians. It implies that risk can be reduced, sometimes substantially, by imposing sign constraints on estimated coefficients, when these signs are prescribed by economic theory. Thus for example, estimated parameters in health effect-pollutant exposure studies should be constrained to be non-negative.

Finally, prior information may be imposed in regression analysis through Bayesian procedures [see Zellner (1971)] which require the selection of prior density functions. The Bayesian procedure, a systematic way of combining sample information with prior information expressed as a density function, minimizes average risk for correct prior densities. However, economists have made little use of this technique because of their general reluctance to specify and test prior densities. The use of priors in model selection is simply a generalization of the procedures of their use in determining functional form and dimension of the design matrix in a single equation context. The use of MLE estimators, pre-test estimators, and Stein-rule estimators to test the validity of restrictions on the parameters in a simultaneous system is totally analogous to their use in a single-equation model. Appropriate restrictions could yield a recursive systems, a system with unobservable variables (but identifiable equations), or a Zellner seemingly-unrelated equation system [see Zellner (1962)] as restrive forms of the general jointly dependent system. Full-in format estimates are consistent and asymptotically efficient. Although single-equation estimators of a simultaneous equation model are biased and inconsistent, they possess minimum variance. In small samples, their risk as measured by mean square error is generally much higher than that of the full- information methods, based on Monte Carlo experiments, even with extremes of multicollinearity, [see Atkinson (1978) and Johnston (1972)]. Thus, the modeller is well-advised to first estimate a simultaneous equation model, if justified by priors, and apply the positive-part Stein-rule estimator to test nested hypotheses on restricted coefficients. Even if incompletely specified, additional restrictions across equations on parameters and, possibly, disturbance covariances aid in identifying the response structure. In addition, when these same cross-equation restrictions are viewed as hypotheses, significance

tests may be used to assess the statistical validity of the model.

Unobserved variables are a special class of errors-in-measurement problems which include omitted explanatory variables, and simultaneous equation systems.

In the air pollution epidemiology literature, attempts to grapple with the measurement error issue have been few. Crocker-Schulze, et al. (1979) raise the simultaneity issue for both air pollution-induced mortality and morbidity. Page and Fellner (1978) employ factor and canonical correlation analysis to attack the unobserved variable problem with respect to air pollution-induced mortality. Otherwise, air pollution epidemiology research largely consists of a vast number or single-equation regressions. Let us briefly examine the relationship between simultaneous equations, unobserved variables, and errors-in-measurement and their impact on estimator risk with the following example. Following Weld and Jureen (1953), who argued that many simultaneous equation relationships involving jointly dependent variables are really recursive relationships, we trace the chain of events from pollutant exposure to behavior change in Figure 1. The outcome at each step in the sequence is conditioned by the outcome in the previous period. Thus, for example, pollution does not immediately affect self-reported disability but rather has a delayed effect via its impact upon metabolism and organ system functions. Consider the following expressions:

$$Y_1 = \alpha_0 + \alpha_1 X_1 + \alpha_2 X_2 + \varepsilon_1, \tag{3}$$

$$Y_2 = \beta_o + \beta_1 Y_1 + \beta_2 X_2 + \varepsilon_2, \tag{4}$$

where Y_1 and Y_2 are, respectively, organ system function and self-reported disability, X_1 is pollution, X_2 is a vector of the other predetermined variables, and the ε's are random disturbances. Given (3), estimating (4) is equivalent to estimating the reduced form equation,

$$Y_2 = \beta_o + \beta_1\alpha_0 + \beta_1\alpha_1 X_1 + \beta_1\alpha_2 X_2 + \beta_2 X_2 + \mu, \tag{5}$$

where $\mu = \varepsilon_2 + \beta_1\varepsilon_1$. If the contemporaneous disturbances in (3) and (4) are uncorrelated, single equation MLE of (3) and (4) are equivalent to full-information estimation of this system.

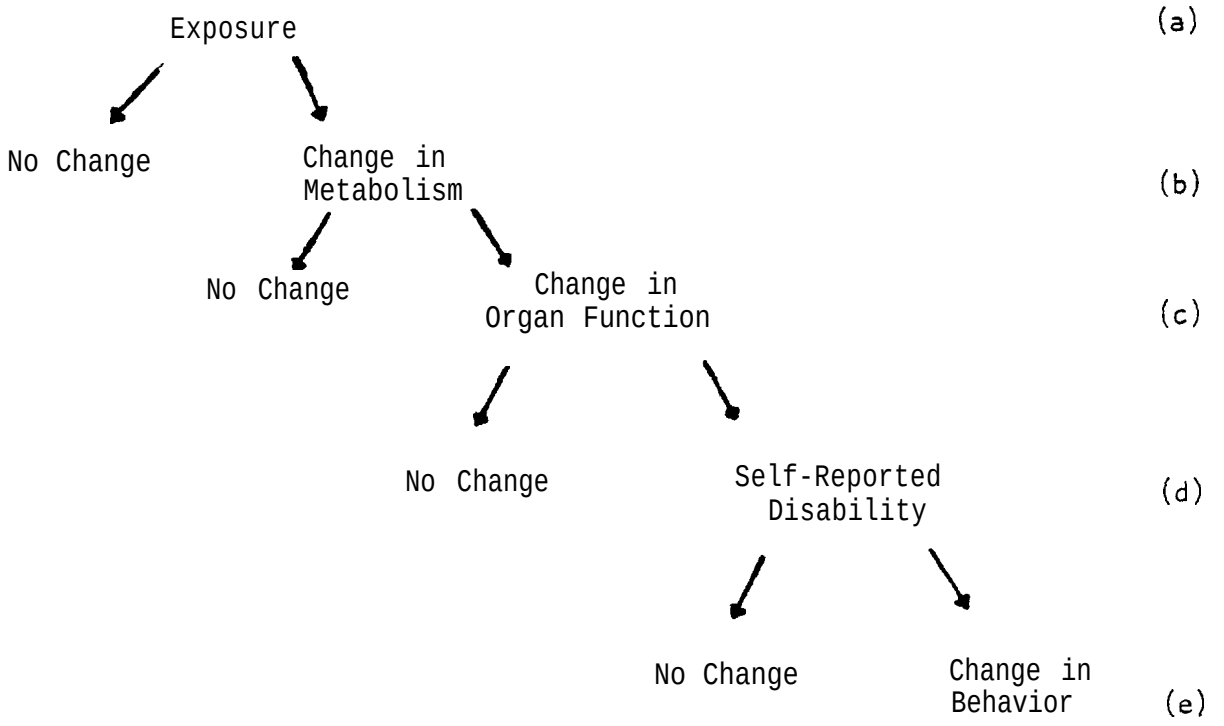
However, if Y_1 is unobservable, some investigators have simply estimated

$$'2 = 'o + \gamma_1 X_1 + \gamma_2 X_2 + \mu \tag{6}$$

Thus, if a MLE of (6) is to yield the same estimate of the impact of

FIGURE 7.1

A SCHEMATIC FOR AIR POLLUTION HEALTH EFFECTS



pollution, X_1, on self-reported disability, Y_2, as would a MLE of (4) given (3), γ_1 must equal $\beta_1 \alpha_1$. For this to occur, ξ_1 and ε_2 and the X's in (5) must be pair-wise uncorrelated [see Judge, et al. (1980, Chap. 13)]. Otherwise the estimate of y_1 will be biased and inefficient.

However, the random disturbances that influence organ system functions seem unlikely to be independent of factors affecting self-reported disabilities. For example, assuming that occupational exposures to toxics is not included among the explanatory variables of (3), and hence are part of the error, an exposure of this sort is likely to intensify the impact in (6) of any particular level of outdoor pollution upon self-reported disability. Instrumental variable methods, which involve the substitution into (4) of a proxy for Y_1 that is both highly correlated with it yet uncorrelated with ε_2, are available to overcome this problem. In the context of the structure represented by (3) and (4), it is not obvious what this proxy might be without additional prior information about (3). Further, use of a proxy in (6) would yield consistent but inefficient estimates of y_1. In short, whether an instrumental variable or a direct measure of X_1 is used, the power of the regression significance tests will most likely be reduced, requiring either a larger sample or more a priori information to maintain a given degree of test power.

Measures of the effective functioning of organ systems completely remove the necessity of wrestling with these particular estimation issues involving unobserved variables. This may be the reason that mortality rates and, more recently, time-to-system failure, have held great appeal as a measure of the health status of a population. Both the biomedical and the economic air pollution epidemiology literature would be considerably advanced through access to direct clinical measures of organ system functions or changes in metabolic processes.

Selection of Values of the Design Matrix

Having selected the appropriate model and the functional form and dimension of the design matrix, additional gains in efficiency can be achieved through the optimal choice of values of the design matrix. This includes both selection of the optimal values of the design variables under the control of the experimenter and the optimal number of observations of each selected value. Solution of this problem [see Figure 1, and Conlisk and Watts (1969)] involves minimizing an objective function, equal to a weighted function of the covariance matrix of the estimated parameters (where weights indicate the a priori importance attached to precise estimation of each variable) subject to a cost constraint on the experiment. The application of this technique to the creation of an epidemiological data base is straight forward. However,

again the estimator risk of this procedure depends on the risk associated with the exclusion of variables from the design matrix, the choice of functional form, and the choice of model to be estimated.

Choice of Density Function for Dependent Variable

The assumed density function of the dependent variable, and hence the error term, has been limited to the normal distribution for purposes of regression analysis throughout the economics literature. However, in many cases, the assumption of a normal density is unwarranted. When the dependent variable is a positive-valued variable representing either time-to-failure for a system or the mortality or morbidity rate for a specific population, previous empirical evidence yields strong priors which argue against the validity of a normal density. In fact, a substantial body of biomedical literature [see Kalbfleisch and Prentice (1980)] has made substantial use of non-normal models. The consequences of incorrectly assuming a normal density are estimator bias, since the parameters describing the likelihood function are incorrect, and possibly a loss in efficiency. Researchers in the biomedical area have adopted two principal models relying on non-normal density functions for the dependent variable in regression analysis. The first involves formulating a parametric regression model based on the generalized F distribution. Parametric restrictions on this distribution specialize it to the Weibull (which further specializes to the exponential), the generalized Gamma (which further specializes to the Gamma), the log logistic, and the log normal [see Kalbfleisch and Prentice (1980)]. Although hypothesis testing for nested densities has been carried out using MLE pre-test estimators, we recommend use of the positive-part Stein-rule estimator for the reasons discussed above. The second principal type of non-normal regression model is the partially parametric Cox (1972) proportional hazards (CPH) model or a non-proportional hazards generalization thereof. The CPH model is termed partially non-parametric because, with the introduction of appropriate parametric restrictions, it specializes to the Weibull and experimental regression models. In the case of a discrete dependent variable, the CPH model specializes to the logistic model. [See Kalbfleisch and Prentice, (1980, pp. 36-37)]. The CPH model has recently been applied to an increasingly wide number of regression problems attempting to explain system time-to-failure. The choice of a partially non-parametric model such as the CPH model in lieu of one of its nested counterparts (e.g., the Weibull or exponential regression models) is again based on minimum risk. Estimated parameters from the CPH model will have less bias that those from the nested models, but will be less efficient. However, Kalbfleisch and Prentice (1980) indicate that the CPH estimator possesses excellent relative asympotic efficiency as well as small sample efficiency compared to nested alterations. Thus, although the evidence regarding efficiency and risk is not

compete, the CPH model appears to afford a considerable increase in flexibility with little increase in risk. Additionally, it allows testing for and accepting its nested densities. The alternative of imposing one of the nested forms appears to offer little gain in efficiency at the risk of considerable increase in bias.

A CRITICAL REVIEW OF THE DOSE-RESPONSE LITERATURE

Over the past decade numerous studies of the economic value of the adverse health effects from air pollution have been carried out by economists and epidemiologists. The ultimate goal of these analyses has been the estimation of defensible functional relationships between dose and response, and then to estimate the resulting economic losses, so that marginal benefits of pollution reduction can be derived from them. The optimal level of pollution control can then be determined where the marginal benefit equals the marginal cost of additional pollution reduction. Recently, substantial controversy has developed over the adequacy and validity of certain methodological approaches and empirical results of studies quantifying dose-response relationships.

In general, there appears to be a minimal attempt in this literature to utilize prior information to formulate and test restrictions of the type previously discussed.

Although the health effects of air pollutants have long been studied in laboratories by toxicologists, there appears to be limited use of this information in non-laboratory studies by epidemiologists. Laboratory experiments on animals allow careful control of the level of individual pollutants, other covariates, and a detailed record of response. These studies, therefore, have been useful for identifying potential human health effects.

Laboratory experiments with human subjects avoid extrapolation from animal to man, but raise other concerns, such as ethical considerations and practical difficulties in studying long-term exposures. In addition, laboratory studies cannot duplicate the activity patterns and pollutant mixture experienced by free-living populations. Within these constraints, experiments involving human subjects can be conducted and used to establish levels at which adverse responses occur after short-term exposures.[27] Despite their limitations, much of what has been learned from laboratory studies could

be employed to provide structure for epidemiological studies. However, many epidemiological studies appear to ignore much of the toxicological literature by assuming linear dose-response functions, thereby failing to investigate possible synergistic effects among pollutants and other important personal factors[3] as well as more complex non-linear mathematical dose-response models based on non-normal distributions, which have been observed by toxicologists.[47]

Studies of occupational groups have been suggested as another source of information. Although such non-experimental studies may allow accurate estimates of exposure, the mix of pollutants and concentrations in workplaces is usually different than the mix in the general ambient air. Exposures are for only work hours rather than the entire day. Temperature and humidity conditions are also likely to differ in important ways from those experienced by the general population. The very young, elderly, and ill are not included. There is considerable selection by the employer and self-selection by the worker, so that those with current disease or those who are more sensitive or more susceptible are found among the employed less frequently than in the general population. Consequently, one cannot extrapolate from findings for occupational groups to the general population. On the other hand, if an association between an air pollutant and a health effect is found in an occupational setting, we would expect a greater association in the general population, if exposed to the same level of the particular pollutant.

In view of these limitations, most of the relevant information about the health effects of air pollutants at levels of exposure near present ambient conditions must come from observational studies of the general population. Here, too, there are limitations with respect to estimating exposure and measuring health effects. Uncontrolled variations in ambient pollution levels make it difficult to determine whether mean concentrations, peak concentration, the variance, or some other measure of air pollution concentration is the most important determinant of health. Additionally, pollution data are usually obtained from outdoor monitoring stations, but the actual exposure burden can vary greatly between individuals even living in the same neighborhood. Outdoor micrometeorology and indoor environment can significantly alter exposure [Benson, et al. , (1972)1 . This imprecision tends to bias estimated associations between air pollution and health effects toward zero. Moreover, health endpoints, including frequency of symptoms, lung function, hospital admissions, and cause of death also are measured with substantial variability. When an association between air pollution and health

is found, a high degree of collinearity between pollutants and the possibility of complex chemical interactions may make it very difficult to associate any health effect with a single pollutant.

Much of the recent work in air pollution epidemiology has focused upon estimation of a linear regression model based on the assumption of a normal error term, where a measure of the incidence of mortality or morbidity is regressed on air quality and other covariates. Many covariates are "personal" factors such as diet, smoking habits, exercise, medical care, age, sex, occupation, income, and genetic predisposition--while others are environmental factors-- such as quality of drinking water, toxic contamination, temperature, humidity, and exposure to allergins.

Many epidemiological studies originating in the biomedical disciplines and sanctified in existing Federal clean air legislation, assumes a positive level of air pollution or threshold below which no individual will suffer a decline in health status.[5/] However, this assumption is clearly a testable hypothesis. The first attempt to employ regression analysis to investigate the health effects of particulate and sulfate air pollution (i.e., principally stationary source pollution) at a national level without the presumption of a threshold was the pathbreaking effort of Lave and Seskin (1970). Using a cross-section of 114 U.S. metropolitan areas, they employed single equation, ordinary-least-squares methods to regress 1960 mortality rates upon ambient concentrations of sulfates and particulate, and other demographic and socio-economic variables. However, they maintained rather than tested the hypothesis that personal factors such as medical care, smoking, and ingestion of fat and alcohol were distributed independently of pollution levels. Thus, Lave-Seskin's analysis is immediately suspected of omitted variable bias, since there is substantial evidence that these factors synergistically interact with air pollution. They tentatively concluded that air pollution caused statistically significant health effects.

This original study has inspired a substantial number of similar studies, including the culminating effort of Lave and Seskin (1977). Included in this list are studies by Gregor (1977), Wyza (1978), Mendelssohn and Orcutt (1979), Seneca and Asch (1979), and Lipfert (1979) involving the mortality effects of sulfur oxides, sulfates, and particulate, and Schwing and McDonald (1976) involving the mortality effects of carbon monoxide, nitrogen dioxide, hydro-carbons, and photochemical oxidants. Studies of the morbidity effects of air pollutants include those by Jaksch (1973) and Seskin (1979). These mortality and morbidity, without exception, all have discerned a significant inverse association between mortality rates and one or more air pollutants, and in general these studies employ the model and functional form of Lave and Seskin. The results of these and more recent studies, which significantly question the

validity of the Lave-Seskin assumptions and results, are summarized in Table 1. V.K. Smith (1977), who used data for 50 U.S. metropolitan areas in 1968-1969, applied versions of the Ramsey (1969) tests for specification error in the general linear model to 36 different single equation specifications. These specifications were similar, and often identical, to those greeted with the most approval by Lave-Seskin, and others. None of the specifications could pass all of the Ramsey tests at the 10 percent level, although four passed all tests except that for non-normal errors which was rejected by all specifications. This result is particularly disturbing. Since Lave-Seskin estimated a linear single-equation model, the change or variable theorem indicates that the dependent variable, mortality rates, are also non-normally distributed. Thus, maximum likelihood techniques should have been employed to estimate a non-normal model, e.g., the Cox proportional hazard model or the Weibull or exponential regression models which are restricted cases thereof. This analysis could even be extended to include Bayesian prior distribution quality and other socio-economic and demographic variables.

Second, Thibodeau, et al. (1980) report on a limited reanalysis of the Lave and Seskin data. While they did notargue the existence of a health-pollution association, they questioned Lave and Seskin's methodology. In particular they found significant lack-of-fit and their reanalysis resulted in estimated effects which differed considerably from those reported by Lave and Seskin.

In a recent monograph, Crocker-Schulze, et al. (1979, pp. 24-71) analyzed 1970 mortality data from a cross-section of 60 cities while trying to correct for potential omitted independent variable and simultaneous equation misspecification. Adding measures of medical care, cigarette consumption, and diet to the single equation Lave-Seskin, specification, they found a nonstatistically significant effect of nitrogen dioxide, total suspended particulate, and sulfur dioxide upon the rate of total mortality,[67] in sharp contrast to the results of Lave and Seskin. Retaining the former variables and accounting for the plausible simultaneity between health status and medical care did nothing to improve the statistical significance of the three air pollution variables. On the presumption that these findings were sufficient to demonstrate the lack of robustness in the Lave-Seskin type results, the authors did not go on to account for the obvious simultaneity between median age (or incidence) and several other plausible sources of simultaneity.

The results of Crocker-Schulze et al. (1979), indicating that the Lave-Seskin type of analysis suffers from omitted variable bias, are given additional support by Graves, Krumm, and Violette (1979) who found significant synergisms between pollutant levels and personal factors in explaining mortality rates. Thus, Lave-Seskin should have tested rather than maintained the hypothesis

TABLE 7.1

A SUMMARY OF EPIDEMIOLOGICAL STUDIES OF AIR POLLUTION

The Effect of Air Pollution on Human Morbidity and Mortality

Mortality

Author	Model and Functional Form	Pollutants Used to Explain Level of Dependent Variable
Lave and Seskin (1970) (1977)	general linear model; linear regression	sulfur oxides and particulate
Crocker et al. (1970) model; linear regression of simultaneous equations	general linear sulfur dioxide and particulate[a]	nitrogen dioxide
Lipfert (1979a) model; linear regression	general linear particulate, and Sulfates[a]	sulfur dioxide
Gregor (1977) model; linear *regression*	general linear particulates[a]	sulfur dioxide
Seneca and Asch (1979) model; linear regression	general linear and sulfur dioxide	sum of particulate
Wyzga (1978) model; linear regression with lagged dependent variable	general linear	particulate

TABLE 7.1 (continued)

Mendelssohn and Orcutt (1979) regression	general linear model, linear	sulfates, carbon monoxide, and sulfur dioxide
Schwing and McDonald (1976)	general linear model; linear regression, ridge regression, and sign constrained least squares	hydrocarbons and nitrates[a]

Morbidity

Author	Model and Functional Form	Pollutants Used to Explain Level of Dependent Variable
Jaksch (1973)	general linear model; linear regression	particulates[a]
Crocker et al. (1979)	general linear model; linear regression and recursive linear regression	nitrogen dioxide sulfur dioxide, and particulate[a]
Graves and Krumm (1979)	general linear model; second order Taylor expansion	sulfur dioxide and particulate
Seskin (1979)	general linear model; linear regression	photochemical oxidant

a Indicates dependent variable explained by personal factors as well as air

that personal factors are independent of air pollution with the framework a simultaneous equation Box-Cox model.

The results obtained by V.K. Smith (1977), Thibodeau, et al. (1980), and Crocker-Schulze, et al. (1979) cast doubt upon the robustness of the Lave-Seskin, et al. estimates, in spite of the no-threshold perspective embodied in these estimates. These doubts are particularly bothersome when the results are extrapolated to project pollution regulation impacts. Nevertheless, before dismissing the hypothesis of an inverse relation between everyday air pollution levels and health states, it must be recognized that Lave-Seskin, et al., may have been asking more of their data than it was capable of giving.[7] Less than one in every 100 people dies in the U.S. each year. No biomedical authority asserts that air pollution is the dominant cause of the deaths that do occur. Many take the view that it is the direct cause of no more than a small fraction of these deaths, although they would agree that it may be quite important in intensifying predispositions toward mortality. However, the general properties of the underlying processes that encourage this predisposition are ill-understood. Thus, even with quite large samples, available estimation techniques and a priori knowledge may be inadequate for distinguishing the mortality effects of air pollution in a human population sample from a host of similar and plausible minor contributing factors.

The possible inadequacy of many available techniques for estimating the existence and/or magnitude of air pollutant-induced mortality applies with special force, given the data Lave-Seskin and their successors had to employ. Their work can be interpreted as an attempt at establishing the probability of a representative individual currently residing in a representative region dying in a given year from a geographically representative level of air pollution. Lave and Seskin justify their use of cross-section regional data on the grounds that these data reflect long-run adjustments by capturing response to pollution levels that have existed for long periods of time. Clearly, this assumption is questionable for many areas where pollution levels and populations at risk (due, e.g., to in and out migration) have changed over time. In addition, since they had no information about the distribution of covariates including air pollution across urban areas, the identifying variabilities of their samples were perhaps drastically reduced.[8] When this relatively low variability of the samples is coupled with what are probably substantial measurement errors in the air pollution variables, attempted corrections in model specification may serve only to misinform.

The preceding remarks lead us to three conclusions. First, given the biomedical and economic subtleties inherent in comprehending the etiologies of air pollution-induced mortality and morbidity, the estimates obtained from aggregated data used in the great bulk of extant studies are unlikely ever to

be sufficiently compelling to establish a consensus. Only when physiological models are coupled with observations on individuals can we expect compelling evidence. Second, statistical power should be substantially increased if research concentrates on morbidity rather than mortality. The frequency, and most likely the identifying variability, of morbidity data appears to be greater than that for mortality data by a factor of fifteen or twenty. Greater variability is also expected with more disaggregated data sets on mortality or mortality for the same reason. Finally, because one's health status is influenced by choices about lifestyles, environmental and occupational exposures to possible toxics, and other health-influencing factors, economics can provide a priori hypotheses and an analytical framework to lend additional structure to epidemiological investigations. The researcher can then further narrow the relationships with which observed real world outcomes can be compared. That is, the limited prior information from the existing epidemiological studies contribute something worthwhile to our goal of parsimonious data collection, but still confronts us with an enormously large parameter space, many elements of which could be insignificant for human health status. The more correct a priori information we can introduce to the problem, the greater the reduction in estimator risk. Given that health effect estimates are to be used for valuation assessments, efforts to reduce the severity of this tradeoff become particularly worthwhile.

A CRITICAL REVIEW OF THE VALUATION OF HEALTH EFFECTS LITERATURE

Economic Valuation of Mortality and Morbidity

Two principal methods of valuing mortality have been utilized in the empirical studies valuing human health. The first involves calculating the discounted present value of earnings lost due to mortality or morbidity [see Weisbrod (1971) and Cooper and Rice (1976)]. This is generally agreed to be an incorrect measure of the true value of mortality and morbidity, whose theoretically correct measure is either the willingness-to-pay to avoid mortality or the compensation required to voluntarily accept such adverse effects.[9/] At best, the discounted present value measure is a very limited estimate of the value of life (e.g., zero for the unemployed or retired) and does not allow for observed trade-offs in the job market between wages and risk of death or injury.

The second method of valuing mortality and morbidity involves estimating willingness-to-pay for risk reduction from: 1) surveys or questionnaires; 2) wage premiums for hazardous occupations; and 3) the cost and estimated effectiveness of safety devices. An individual's willingness-to-pay for a small reduction in the probability of death is generally extrapolated to

calculate the value of statistical life.

Two willingness-to-pay surveys have been conducted to estimate the value of life. Acton (1973) asked a sample of 37 Boston area residents to state their willingness to pay for emergency coronary care facilities which would reduce the probability of a fatal heart attack. From the responses, Acton estimated a value of life of less than $100,000 ($ 1978). Jones-Lee (1976) estimated a far higher value of life in excess of $6 million ($ 1978) for safer air travel, by asking travelers their willingness to pay higher fares to travel on airlines with lower probabilities of a fatal crash. However, difficulties in obtaining reliable estimates to theoretical questions arise because of incentives for strategic behavior, e.g., with public goods, and the limited ability of the individual to make an accurate determination of preferences in hypothetical situations. See Freeman (1979) for a discussion of attempts to overcome various types of strategic bias.

A more fruitful approach has been taken by a number of studies attempting to measure the value of life from data on wage differentials in hazardous occupations. Thaler and Rosen (1976) analyzed a sample of 900 individuals in 37 high-risk occupations taken from the records of the Survey of Economic Opportunity. They explained wage differentials among these occupations with: (1) the extent to which the risk of accidental death exceeded the expected average from statistical life tables; (2) regional and urban dummy variables; (3) demographic characteristics; and (4) job characteristic and occupational dummy variables. By extrapolating risk to zero, Thaler and Rosen calculated a value of life ranging from $273,000 to $508,000, with a best estimate of $391,000 ($ 1978). Using the same data on wages but different estimations of occupational risk, R.S. Smith (1976) obtained substantially higher estimates of the value of life, ranging from $2.2 million to $5.1 million ($ 1978). Finally, using a different data set, Viscusi (1976) obtained estimates ranging from $1.8 to $2.7 million ($ 1978) for blue-collar workers.

Three caveats must be applied to the use of these estimates. First, they represent the value of marginal changes in the probability of death extrapolated to a zero probability of death. If the marginal valuation of different probabilities varies significantly, this extrapolation may be highly biased. Secondly, the willingness to pay measured by these studies most likely is associated with accidental death and excludes the value of the disutility associated with the morbidity, pain, suffering with characterize fatal but chronic diseases such as cancer. Thus, these estimates may understate the willingness to pay by the general population. Finally, data on risk by occupation are not corrected for the fact that omitted personal characteristics are often associated with high risk jobs which account for non-job related deaths. Thus, a certain component of increased mortality

cannot be associated with a corresponding wage differential.

Studies estimating the willingness to pay by the general population for risk reduction as evidenced in consumer purchases of safety devices include those by Blomquist (1979) and Dardis (1980). Blomquist (1979) developed a simple life-cycle model of individual life-saving activity and estimates a value of life based on automobile seat belt use. Solution of his simple utility optimization model yields the first-order condition that the marginal value product of reduced mortality plus the marginal value product of reduced morbidity equals marginal cost. Blomquist then used probit analysis to explain the incidence of seat belt use with a set of demographic variables, length of work trip, speed limit, labor wealth, and wage rate. This fitted equation, evaluated at the mean of the data is equated to the net marginal benefits of seat belt use, up to a factor or proportionality, equal to the variance of the dependent variable. Assuming zero time and disutility costs of operation, the implied value of life is solved from this equation. His estimates of the average value of life, based on a non-random sample of about 5,500 households in A Panel Study of Income Dynamics, 1968-1974 is $370,000 ($ 1978) . However, Blomquist relies heavily of the estimated wage coefficient in the profit equation to estimate the variance of the dependent variable. To the extent that the wage rate does not accurately reflect value of life, these estimates will be biased.

Dardis estimates willingness to pay for risk reduction by examining data on consumers' voluntary purchase of smoke detectors and their expected reduction in the incidence of death by fire. He estimates the annualized cost of smoke detectors per household based on a catalog purchase price, life expectancy of ten years, an average of 1.5 smoke detectors per household, and discount rates of 5% and 10%. Then under the assumption that 13% of households in 1976 were equipped with detectors, that only 80% of these were functional, and that these functional detectors provided only 45% protection, the total deaths in the absence of functional detectors was estimated at 6,492. Savings of life from the provision of smoke detectors in each household was then estimated at 2,337 (equal to .8 x .45 x 6,492) for a probability of reduction in death of 3.16×10^{-5} for all households. Combining this probability with the annualized cost of smoke detectors yielded estimates of the value of life to purchasing households ranging from $293,000 to $341,000 ($ 1978). The estimated value of life to the entire population was considerably less - ranging from $157,000 to $175,000 ($ 1978).

Although the behavior of the general population is observed in these two studies of consumer safety devices, there are many important shortcomings to their work. The first two caveats associated with the wage rate willingness to pay studies also apply to the studies by Blomquist and Dardis. In

addition, the most serious problem with Dardis' approach is that the total value of consumer willingness to pay cannot be accurately estimated using the selling price of the safety device. Clearly, many consumers with higher subjective probabilities of risk would pay far more than the modest price of the detector, whose production costs are substantially lowered by scale economies. However, the empirical importance of this bias is not clear. In light of these shortcomings, we suggest the following theoretical structure for hypothesis testing in valuing health effects.

The problem of valuing health effects is the discovery of the rates at which individuals are willing to substitute air pollution-induced changes in health status for money or its equivalent. The conceptual framework employed in the great bulk of the work on the demand for health is the household production model, particularly its human capital versions [Grossman (1972), Crocker-Schulze, et al. (1979, pp. 137-149)]. In this framework, the individual or family unit is viewed as a firm attempting to maximize utility subject to constraints on the household budget and the production of goods and services which yield utility. Market goods and services are purchased and combined with the time of various family members in production. Household members are therefore implicit demanders of their own time resources as well as of the factors, including health status, that influence what they are able to do with these time resources. The framework is useful for studying the value of air pollution-induced health effects because: (1) it assesses individual well- being by "full income"--the value of all the individual's time, including time passed in productive nonmarket activities such as raising children--and not merely by his money income; and (2) it provides a means of introducing a priori information on behavior of organ systems into a health production function.

Within the household production framework, changes in behavior due to a change in air pollution-induced health status flow from three major sources. First, a change in health status can change the income and wealth positions of some individuals, thus changing the amount and possibly the mix of "commodities" these individuals consume. Second, changes in health status may influence the type of income sought by the individual. Individuals can be expected to shift their efforts and investment patterns toward obtaining those types of income that yield the highest net return for expended time and money. Alternatively, because of increases in the difficulty of internal financing, reductions in self-investment, job search schooling, on-the-job-training, and migration may occur. Finally, various income support programs as well as the individual's social reputation are contingent upon others' perceptions of one's health status. Therefore, to the extent possible, individuals will tend to tailor their self-reported health status to increase their chances of being categorized in a manner offering them the most advantageous time and money

terms.

Thus, changes in wage rates and income will reflect, to a degree, changes in health status. Wages, which are the most important source of income for most households, are fairly accurately reported in most data sets. However, this by no means implies that they are free of measurement error and other problems. There are" at least three major difficulties with most wage data.

First, the individual's behavior is based upon his marginal, not his average, wage rate. The marginal wage rate is net of taxes and it must be adjusted for fringe benefits and for the cost-of-living. Since marginal and average rates obviously differ for all persons subject to progressive income taxes, failure to take account of these taxes will bias toward zero the estimated coefficient relating hours worked to wages.

Second, the wage rate used for estimation should distinguish between the permanent and the transitory components of wages [J.P. Smith (1977)]. The observed wage rate may be systematically related to the wages the individual expects to receive in the future. Ignoring anticipations regarding wage profiles over the life cycle can lead to seriously biased results. For example, if people who currently receive relatively high wages anticipate more steeply sloped wage profiles than do low wage people, the effect of current wage on labor supply is likely to be underestimated. To help control for the effect of differences in permanent and transitory wages, J.P. Smith suggests estimating expressions using cross-sectional data on narrowly defined age groups.

Third, data must be provided that allows the imputation of wage rates for nonworkers, many of whom adopt this status because of health problems. One solution is to impute a potential wage rate for nonworkers on the basis of the wages observed for healthy persons of otherwise similar characteristics for whom wage data is available. Gronau (1974) shows, however, that this procedure will overstate wage rates for individuals belonging to groups with low labor market participation rates.

Changes in income can occur for reasons other than changes in the wage rate. In particular, it is necessary to know the individual's and the household's nonemployment income flows. For most households, the primary sources of nonemployment income are the home and the automobile. Ignoring the nonmonetized returns from these assets can seriously bias estimated relations between changes in income levels and changes in behavior. J.P. Smith (1977) suggests that the problem of imputing values to nonmonetized assets can be avoided if subsamples are defined to include individuals who are at the same point in their life cycles and have had similar wage paths and other factors

that may influence their time allocations over the life cycle.

Another important determinant of individual income is the amount of labor the individual supplies. Because of disequilibria in labor markets, the actual hours of employment for some persons may differ substantially from the number of hours they wish to work at the wage rate they receive [Ashenfelter (1977)]. When assessing the value of air pollution-induced changes in health status, we wish to know the changes in actual hours worked.

All of the above wage and labor supply responses may differ among various types of people; that is, the characteristics defining types of people may interact with the explanatory variables of the expressions to be estimated. When these characteristics are exogenous, and when the existence but not the form of the interaction is known, the sample must be stratified so that separate estimates can be made for each type. Failure to do so can lead to seriously misleading estimates. Crocker and Horst (forthcoming) have shown, for example, that reductions of the earnings of workers in the same occupation exposed to near-identical ambient concentrations in Los Angeles vary between zero and nine percent. Pooling these workers would have imposed statistically unacceptable restrictions. In light of the preceding discussion of the optimal use of prior information we draw the following conclusion: in the absence of prior information and hypothesis testing, the "ideal" data set cannot be specified. One can only say that data on all imaginable factors that affect health status, will not be ideal since it will produce intolerable risk. To minimize risk, we must introduce priors from accumulated statistical evidence to structure testable hypotheses about functional form; dimensionality of the parameter space, the model, the values of the design matrix under experimental control, and the density of the dependent variable, and we must employ optimal test procedures otherwise, there is no optimal way to judge the value of a data set. A good approximate specification of what would be ideal must therefore wait upon the results of explorations of what is gained by imposing more structure on existing data sets. For example, the introduction into the model structure of expressions for metabolic processes and organ system functions can provide identifying restrictions for the parameters of the self-reported disability, even though such data are scarce. Of course, the most complete identifying restrictions would be obtained if direct observations were available on these processes and functions. A data set having these observations could then be used to assess the gains from including expressions for these undeserveable processes and functions in the model structure relative to the gains from having direct observations on them. Given the likely expense of collecting accurate data on organ system functions, for example, a prior assessment of the size of these gains seems a worthwhile investment.

steps in the causal chain in Figure 1 as the HES data set. This is the Health Examination Survey (HES) data set collected from late 1959 through 1962 for a nationwide sample of 7710 adult, civilian, noninstitutionalized individuals [National Center for Health Statistics (1965)]. Given the early date of the HES data set and the broadness of its locational information (counties or sets of counties), more measurement error than usual would be introduced when the set was matched with air pollution information. However, as Leaderer (1979) has suggested, visibility information from airports might serve as a very adequate proxy for fine particles which are suspected as the major source of health impairment from air pollution.

CONCLUSIONS AND RECOMMENDATIONS

Neither epidemiologists nor economists are yet able to provide estimates of the health consequences of air pollution with sufficiently reliable hypotheses to carry out a defensible cost-benefits analysis. The range of uncertainty is unacceptably large. A traditional response to unacceptably large ranges of uncertainty is a plea for undertaking a fresh data collection effort. To say that one wants all "feasible" information on individuals' genetic and social endowments, metabolic processes, organ system functions, past and present life-style habits, risk exposures other than air pollution, attitudinal variables related to stress, indoor and outdoor air pollution exposures, family characteristics and employment opportunities, as well as a history of time and budget allocations is to say little. Minimization of estimator risk requires physiological and economic models to specify testable hypotheses and hence to guide the data specification. A great deal of relevant economic information will have been made available when the measures of labor supply, wages, and income described in the previous sections are generated. Smoking habit information, diet, and occupational exposures appear to be necessary. Beyond this, data sets must be collected and explored with the explicit objective of minimizing estimator form, model selection, experimental control of the design matrix, and choice of density function for the dependent variable. This will require that more attention be devoted to the role played by organ system functions using data disaggregated to the individual level. Expressions which purport to explain these functions, along with expressions which explain time and budget allocations, will most likely become the major sources of a *priori* information that can be used to bound the investigation. Thus, the epidemiologist is at the difficult position where more testable hypotheses appear to be as important as more data.